Results There are 242 chemical substances included in the Portuguese NHFM that were classified as PIM by at least one of the three tools. It was observed that, of these 242 chemical substances, 181 were classified as PIM by the STOPP criteria, 136 by the EU(7)-PIM list and 64 by Beers criteria. About 17% of identified PIMs were present in all three tools. About 27% of all PIM in the NHFM belonged to the ATC group C (cardiovascular system), 23% to group N (nervous system) and about 15% to group A (alimentary tract and metabolism). The SmPC of about 36% of the identified PIMs did not have special recommendations or precautions for use in older patients.
Conclusion and relevance Identification of PIM by hospital pharmacists, using adequate tools, is essential to contribute to the reduction in drug related problems in older patients.

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No conflict of interest.

5PSQ-016 TOLVAPTAN ASSOCIATED CREATINE KINASE ELEVATION IN TWO PATIENTS WITH AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

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Background and importance Autosomal dominant polycystic kidney disease (ADPKD) is a genetic disorder that causes kidney damage; the treatment goal is to postpone renal failure. The only specific treatment approved for ADPKD is tolvaptan (Jinarc, Otsuka Pharmaceutical), an arginine–vasopressin receptor antagonist taken orally—45 mg in the morning and 15 mg in the evening.
Aim and objectives To present two cases of tolvaptan associated toxicity.
Material and methods The cases were detected and monitored by a nephrologist during outpatient visits in our centre, and laboratory tests were done during this time. After a suspicion of tolvaptan associated toxicity, the electronic clinical records and laboratory tests were reviewed.
Results Case 1: a 43-year-old man with ADPKD, whose mother had ADPKD, had been using losartan, carbonate calcic, manidipine, valsartan and hydrochlorothiazide. He started tolvaptan at the lowest dose. It was well tolerated and weeks later creatine kinase (CK) plasma levels increased dramatically (table 1). Tolvaptan was stopped and CK levels recovered to baseline levels. The patient reported he felt better after treatment discontinuation.

Case 2: a 41-year-old man with ADPKD, whose mother and siblings were also affected, was treated with enalapril, amlodipine and allopurinol. He started tolvaptan at the lowest dose with good tolerance. An increase in CK was detected, treatment was stopped (all other treatments continued) and CK plasma levels declined (table 1).

Abstract 5PSQ-016 Table1 Evolution of CK and creatinine plasma concentrations

	Date	Treatment duration (days) (*days after treatment cessation)	CK levels (UI/L) (55–171 UI/L)	Creatinine (mg/dL)
Patient No 1	11/12/2018	11	264	1.73
	19/12/2018	19	585	1.74
	27/12/2018	*7	356	1.72
	09/01/2019	*20	278	1.8
	15/02/2019	*36	244	1.64
	13/03/2019	*65	308	1.88
	17/03/2019	*69	312	1.82
	29/05/2019	*161	248	1.82
Patient No 2	10/05/2019	5	153	1.62
	22/05/2019	17	854	1.65
	24/05/2019	*1	712	1.65
	30/05/2019	*8	304	1.76
	05/06/2019	*13	358	1.72
	30/06/2019	*28	167	1.58

Neither patient No 1 nor patient No 2 showed clinical symptoms. They reported that they had not taken other treatments and had occasionally performed moderate exercise, as usual. In the absence of other justifications, according to the *Naranjo* causality assessment, it was probable (6 points) that tolvaptan caused hyperCKaemia.
Conclusion and relevance These are the first cases of tolvaptan induced hyperCKaemia reported. HyperCKaemia could be common in ADPKD patients taking tolvaptan and might be underestimated. It is advisable to monitor CK serum concentrations in these patients.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

5PSQ-017 PCSK-9 INHIBITORS: REAL WORLD EFFECTIVENESS

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Background and importance Pharmacological therapy for hypercholesterolaemia aims to reduce circulating low density lipoprotein (LDL) concentrations. A new therapy for patients who fail to achieve the desired targets consists of monoclonal antibodies that selectively and irreversibly bind proprotein convertase subtilisin/kexin type 9 (PCSK9) to prevent its binding to the LDL receptor (LDL-R)/LDL complex on the surface of hepatocytes. Increased LDL-R liver levels result in serum reduction of LDL cholesterol.
Aim and objectives The aim of this study was to define the effectiveness of two inhibitors, alirocumab and evolocumab, using changes in lipid parameters and ratios of patients during therapy. Furthermore, an additional goal was calculation of the 10 year cardiovascular risk according to the Framingham Heart Study algorithm that includes age, sex, systolic pressure, smoking, diabetes, antihypertensive therapy, LDL, high density lipoprotein (HDL) and total cholesterol.
Material and methods The study was conducted from May 2017 to September 2018. The 120 enrolled patients had at least a 6 month re-evaluation. Data were extracted from the